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ABSTRACT

Reported was an explanatory conceptual model for pathological left-handedness (PLH) and related hypotheses, some of which could not be tested empirically due to lack of information. The model was reported to provide an explanation for the relationship between handedness and specific learning disability, and handedness and cerebral dominance for speech. The model stated that the incidence of PLH will increase as a function of early brain injury. It was demonstrated that the ratio of PLH to pathological right-handedness was independent of the magnitude of the value of manual switch. The two hypotheses which could not be tested involved the manual switch value and the relationship of the incidence of familial sinistrality, in pathological left-handedness groups, to that in normal right-handed control groups. Hypotheses said to be tentatively proven focused on the probability of brain lesion in the left hemisphere if the subject is left-handed and is retarded or epileptic, the probability that such a person is a pathological left-hander, the incidence of manifest left-handedness in brain-injured populations with perinatal or early post-natal injury, and the incidence of pathological left-handedness in comparison to that of natural left-handedness. (CB)

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Pathological Left-Handedness
An Explanatory Model[1]

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For over seven decades investigators have reported a raised incidence of manifest left-handedness in a variety of clinical populations, including stutterers and disabled learners (Hildreth, 1949; Zangwill, 1962; Palmer, 1964) and, in particular, mental retardates and epileptics (Bingley, 1958; Hecaen & Ajuriaguerra, 1964). The two latter groups are of special interest since incidence rates and etiology are more often reported. Briefly, investigators have consistently reported an incidence of manifest left-handedness of approximately 17 percent in both mentally retarded and epileptic groups, primarily children (Mayet, 1902; Redlich, 1908; Steiner, 1911; Steir, 1911: Gordon, 1920; Hildreth, 1949; Trankell, 1950; Hecaen & Piercy, 1956). Hildreth (1949) and Bingley (1958) stated that this incidence rate represents at least a twofold increase over that reported for normal control children (approximately eight percent).

The explanation for this increase in manifest left-handedness, in brain-injured groups, postulates that damage to the left hemisphere causes a mild hypofunction of the contralateral hand, in natural right-handers, which in turn causes the child to switch to the opposite hand for manual activities. Redlich (1908) and Bingley (1958) concur that such cases of manual transfer are examples of pathological left-handedness (PLH) which should be differentiated from natural or inheritable left-handedness (NLH).

This explanation, while untested and rarely contested (Steiner, 1911), has been tacitly accepted by neurologists and psychologists for decades.

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Closer examination of this hypothesis, however, raises a number of questions. First, if a phenomenon such as pathological left-handedness exists, then what about the converse effect--namely, pathological right-handedness? Although Bingley (1958) mentions this possibility, its occurrence and explanation is dismissed. Nevertheless, if brain lesions are random in nature, then there must be cases of right hemisphere damage in natural left-handers who, by virtue of hypofunction of the contralateral hand, are predisposed to become pathological right-handers.

Second, what is the explanation for the consistent reports of a 17 percent incidence rate, if true, in epileptic and brain-injured populations? Stated in another way, no one has yet determined the probability of manual transfer (switch) which would account for the twofold increase in manifest left-handedness in brain-injured populations. It would be preposterous to asuume, on clinical grounds, that all brain lesions contralateral to the natural or dominant hand would result in manual transfer. Even Hecaen, et al. (1964) have reported cases of infantile right hemiparesis in children who retained their preference for the right hand in spite of severe contralateral motor deficit.

Third, what explanation would account for the more consistent reports of a raised incidence of manifest left-handedness in retarded and epileptic groups as opposed to other clinical groups (e.g., stutterers, disabled learners, brain-injured adults)? Although incidence studies in retarded and epileptic groups have largely been comprised of children (Hildreth, 1949), the fact remains that similar age groups have been represented in cases of stuttering and specific learning disability (Palmer, 1964). Therefore, age by itself may not represent a sufficient explanation for this twofold increase in left-handedness in retarded and epileptic groups.

The purpose of the present paper is to offer a model which attempts to answer each of the preceding questions and which generates a number of testable hypotheses, some of which are logically derived from the proposed model. The hypotheses are assumed to have diagnostic utility and are empirically tested in the present paper.

The initial part of the model is presented in Table 1 for 1000 hypothetical cases who are assigned to a 2 x 2 array of natural hand side (L, R) and potential brain lesion side (L, R). Table 1 therefore generates the distribution of handedness by lesion side in which left-handedness is estimated at eight percent (Hecaen & Ajuriaguerra, 1964) and lesion side is estimated at 50 percent (random occurrence in nature).

On the basis of these initial assumptions, it is then necessary to determine the probability of manual transfer, given a random unilateral lesion, which would alter natural hand preference by increasing manifest left-handedness to around 17 percent. This determination is possible by the algebraic solution for an unknown event which, in Table 1, would refer to that proportion of natural right-handers (N = 460) who, after left brain damage, would become pathological left-handers (PLH), and that proportion of natural left-handers (N = 40) who, after right brain damage, would become pathological right-handers (PRH) such that the incidence of manifest left-handedness would result in 170 cases (17 percent). The equation is expressed as follows:

$$\begin{aligned} 40 + 460x + 40 - 40x &= 170 \\ 80 + 420x &= 170 \\ 420x &= 90 \\ x &= .21 \end{aligned} \qquad (1)$$

This solution indicates that a probability of p = .21 representes the estimated value of manual switch (P_{SH}) which would produce a manifest inci-

dence of 17 percent left-handers in retarded and epileptic groups.

Table 2 illustrates this redistribution of cases, given a random brain lesion, where P_{SH} = .21. Inspection of the table indicates that there would be 40(N) + 97(P) + 32(N) = 169 manifest left-handers (17 percent).

It is also obvious from Table 2 that the number of pathological left-handers (N = 97) is far in excess of pathological right-handers (N = 3). The ratio is approximately 11.5:1 which is almost identical to the ratio of natural right-handedness (N = 920) to natural left-handedness (N = 80) [ref. Table 1]. The model (Table 2) thus explains the relationship between pathological left- (PLH) and right-handedness (PRH). The ratio (11.5:1) is identical to the ratio between natural right-handedness and left-handedness (11.5:1). Therefore, the reason that pathological right-handedness is seldom discussed is that its true frequency is perforce rare. It is restricted by the lower frequency of natural left-handedness in the population.

It can also be demonstrated that this ratio between PLH:PRH will hold for conditions in which P_{SH} varies. In other words, if one assumes that P_{SH} = .50, then inspection of Table 3 shows that the ratio between PLH and PRH is again approximately 11.5:1 (230:20) which proves that the ratio is independent of the magnitude of the probability for manual transfer (P_{SH}).

It is also evident from Table 3 that the assumed value of manual switch (P_{SH} = .50) would produce a much larger number of manifest left-handers [40 (N) + 230 (P) + 20 (N) = 290 or 29 percent] than that estimated from the algebraic equation (1). This fact merely suggests that the incidence of manual switch, after contralateral brain-injury, has a low probability of occurrence in nature (P_{SH} = .21).

Thus far, the model explains the relationship between pathological left-handedness and pathological right-handedness (Hypothesis 1). In addition, the

model estimates the probability of manual transfer (switch), given a lesion in the hemisphere contralateral to the natural hand, which fits the empirical data on the raised incidence of manifest left-handedness in retarded and epileptic populations ($P_{SH} = .21$).

Assuming that this estimated value is correct ($P_{SH} = .21$), two additional hypotheses can be logically derived from the model (Table 2).

Hypothesis 2. If a subject is left-handed (LH) and is retarded or epileptic (BD), the probability (P) that the primary lesion is in the left hemisphere is:

$$P(LB|LH + BD) = f\ LB/f\ (LB + RB) = 40(LB) + 97(LB) = 137/169 = .81 \quad (2).$$

That is, approximately four out of every five retarded or epileptic Ss, who are manifest sinistrals, should have a primary lesion in the left hemisphere.

Hypothesis 3. If a retarded or epileptic subject is left-handed (LH) and has a left hemisphere lesion (LB), the probability (P) that he is a pathological left-hander (PLH) is:

$$P(PLH|LH + LB) = f\ PLH/f\ (PLH + NLH) = 97/137 = .71 \quad (3).$$

The following hypotheses are only indirectly related to the model. They do not represent logical deductions as such. Nevertheless, they are testable, and if true, would provide greater specification for some of the variables in the model (e.g., P_{SH} and PLH).

Hypothesis 4. The incidence of manifest left-handedness will be raised primarily in brain-injured populations with perinatal or early post-natal injury (e.g., epilepsy and mental retardation). This hypothesis is based on the assumption that the likelihood of transfer in manual functions, due to contralateral brain injury, is greater when the nervous system is more plastic and is subject to more rapid growth (Lenneberg, 1967). This hypothe-

sis fits with other observations which indicate a raised incidence of manifest left-handedness, particularly in younger brain-injured age groups (Bingley, 1958; Hecaen et al., 1964).

Hypothesis 5. If the incidence of manifest left-handedness is 17 percent in brain-injured populations, particularly retarded and epileptic groups, then there should be at least one pathological left-hander (PLH) for every natural left-hander (NLH). Thus, P(PLH) ≧ P(NLH) or R(PLH) : (NLH) = 1:1. This hypothesis is based on the fact that an incidence rate of 17 percent represents a twofold increase over the rate of natural left-handedness in normal populations. Thus, the ratio of PLH : NLH should be approximately the same.

Hypothesis 6. The incidence of familial sinistrality, in pathological left-handedness groups, should be similar to normal right-handed control groups. Or, the incidence of familial sinistrality should be higher in normal left-handed children than in brain-injured left-handed children (e.g., epileptic).

This hypothesis is based on the fact that pathological left-handers are intrinsically natural right-handers who have transferred manual preference and hence should have the same familial handedness as natural right-handers. Some indirect support for this hypothesis has already been reported by Redlich (1908) and reviewed by Bingley (1958).

TEST OF THE MODEL

Although each of the predictions from the model are testable, there are few large-scale studies in the literature that would satisfy some of the assumptions implicit to the model. First, the study would have to be based on a fairly large and representative sample of brain lesion cases in which lesion specificity was rigorously determined. Second, the brain lesion cases should be comprised of mental retardates, or, preferably, epileptics. Third,

data should be available on approximate age of brain injury in order to differentiate the effects of early versus late-occurring lesions. Fourth, the distribution of lesion laterality should be approximately random. Fifth, classification of handedness should be based on some systematic questionnaire or dexterity measure.

After considerable search it became apparent that one particular study satisfied the preceding assumptions--namely, the Montreal Neurological Institute data on handedness and cerebral dominance (Penfield & Roberts, 1959). The chapter by Roberts (pp. 89-102) presented data on the relationship between aphasia and handedness, in 522 epileptic patients, after unilateral lobectomy. If one assumes that the operation was performed on the side of the epileptogenic lesion and one excludes the data on aphasia sequelae, then Roberts' data can be rearranged as in Tables 4 and 5 in the present paper.

Inspection of Table 4 shows that the side of the epileptogenic lesion was approximately random in the 522 cases, although slightly higher on the right (53 percent). Table 4 also reveals that the incidence of manifest left-handedness (17 percent) was identical to previous reports of sinistrality in epileptic populations (Bingley, 1958). The data in this table now represent an empirical test for some of the hypotheses derived from the model in Table 2.

Hypothesis 2. $P(LB \mid LH + BD) = f\ LB/f\ (LB + RB) = .81$ (Table 2). Inspection of Table 4 reveals that 67/89 or 75 percent of the left-handed epileptic patients (BD) had their primary lesion in the left hemisphere. This observed percentage closely approximates the value of 81 percent predicted by the model (Table 2).

Hypothesis 3. $P(PLH \mid LH + LB) = f\ PLH/f\ (PLH + NLH) = .71$ (Table 2). In order to test this hypothesis, it is necessary to inspect Tables 4 and 5. The data in Table 5 are similar to Table 4 except for the removal of those

patients who sustained brain-injury before the age of two (Penfield & Roberts, 1959, p. 93). By removing Ss with early brain injury, it reduced the frequency in the left-hand, left-brain cell (LB) from a total of N = 67 (Table 4) to N = 18 (Table 5). If one assumes that these excluded cases were pathological left-handers (67 - 18 = 49), then $P(PLH \mid LH + LB) = 49/67 = .73$.[3] This observed value again closely approximates the value of 71 percent predicted by the model in Table 2.

Hypothesis 4. The prediction that the incidence of manifest left-handedness will be raised primarily in brain-injury populations with perinatal or early post-natal injury (e.g., epilepsy and mental retardation) is indirectly supported by Penfield & Roberts' data (Tables 4 and 5). After removal of those patients with brain injury prior to age two, the incidence of manifest left-handedness dropped from 17 percent (Table 4) to eight percent (Table 5), which approximates the incidence rate for the normal population (Table 1).

Hypothesis 5. $P(PLH) \geq P(NLH)$ or R(PLH) : (NLH) = 1:1. This hypothesis merely estimates the ratio between pathological and natural left-handedness, given that the incidence of manifest left-handedness is 17 percent. The hypothesis predicts that there should be at least one PLH for every NLH in childhood brain-injured groups. Inspection of Tables 4 and 5 indicates that there were approximately 49 (67 - 18 = 49) PLHs out of 522 Ss (9 percent), and approximately 33 (18 + 15 = 33) NLHs out of 386 Ss (8 percent) after removal of the early brain injury cases (Table 5). This ratio between pathological left-handedness and natural left-handedness can be visualized more easily by observing the twofold decrease in manifest left-handedness (17 - 8 percent) after removal of the early brain lesion patients.

DISCUSSION AND CONCLUSIONS

Two hypotheses (H_1 and H_6) could not be tested empirically in the present study because certain additional information was not reported in the Penfield & Roberts (1959) study. The ratio of pathological left-handedness (PLH) to pathological right-handedness (PRH) could not be evaluated because the patients were not classified on this dimension (Hypothesis 1). One might argue, however, that this hypothesis could be proved as a logical or mathematical deduction from the model (Tables 2 and 3). Briefly, the ratio of PLH : PRH should be identical to the ratio of NRH : NLH (i.e., 11.5:1). Thus, the low occurrence of pathological right-handedness should be restricted by the low base rate incidence of natural left-handedness in the population. Moreover, it was demonstrated that the ratio (PLH : PRH) was independent of the magnitude of the value P_{SH} (i.e., manual switch). Nevertheless, the lack of direct information on PLH could render the test of Hypothesis 3 $[P(PLH|LH + LB)]$ invalid. This criticism is justified and should warrant caution in accepting the results of the test for Hypothesis 3.

Hypothesis 6 could not be tested because no information was available on familial handedness. Although indirect support for this hypothesis has already been reported by Redlich (1908), the validation of this prediction is not felt to be central to the model. Nevertheless, it would be interesting to see whether the incidence of familial sinistrality was similar in both pathological left-handers and normal right-handers. Trankell (1950) has shown that the probability of left-handed kin is at least doubled in left-handed offspring (52 percent) compared with right-handed offspring (23 percent).

The remaining hypotheses were in essential agreement with the observed data (Tables 4 and 5). The model could therefore be accepted tentatively until additional empirical data are collected or experimental studies are undertaken.

Although the model has heuristic value, its primary value seems to rest on the hypotheses, which are clearly testable, and the explanations which are offered on the phenomenon of left-handedness. The model states that at least one out of every two manifest left-handers in retarded and epileptic groups are pathological left-handers. Moreover, if the S is a manifest left-hander, the probability is very high ($p = .81$) that the primary lesion is in the left hemisphere, regardless of whether he is a pathological left-hander or natural left-hander. These predictions thus provide useful diagnostic information for the clinician.

The model, however, provides a more useful explanation for the investigator who has long tried to interpret the controversial (if not muddled) results on the relationship between handedness and specific learning disability, and handedness and cerebral dominance for speech (Zangwill, 1962; Sparrow and Satz, 1970). The model states that the incidence of pathological left-handedness will increase as a function of early brain injury. Consequently, if studies of learning disabled children are carried out in medical settings where the incidence of brain damage is higher, then the chances should also increase for selecting a pathological left-hander which would spuriously increase the chance of finding a relationship between handedness and specific learning disability. Conversely, if similar studies are carried out on learning disabled children in public schools, the incidence of brain damage and pathological left-handedness should both be lower which should attentuate or wash out any relationship between handedness and the criterion variable. Recent reports have tended to confirm this observation on sampling selection (Belmont & Birch, 1966; Sparrow & Satz, 1970). If true, it would help to explain the relationship between handedness and childhood learning disability as an artifact of sampling procedure or pathological left-handedness.

Similarly, studies addressed to the relationship between hand preference and

cerebral dominance for speech would be subject to the same criticisms. This would be particularly true in the above case because the majority of such studies are based on brain-injured patients who are generally epileptic. Branch, Milner & Rasmussen (1964) have already shown that the relationship between manifest left-handedness and hemispheric speech dominance, using the Sodium Amytal procedure, varies dramatically as a function of the age of the lesion. The majority of left-handed patients, with evidence of early left brain damage, showed contralateral speech dominance on the right, whereas the converse relationship obtained for those left-handed patients with no evidence of early left-brain damage (p. 403). In other words, the former patients (early left brain disease) were undoubtedly pathological left-handers (PLH) who, if not excluded from the natural left-handers (NLH), would have produced a more variable and erroneous estimate of the relationship between left-handedness and hemispheric speech dominance. Unfortunately, most of the research in this area has been based on brain-injured patients for whom information was not available on age of the lesion. Consequently, the conclusions advanced from these studies must be questioned. The model suggests that the contribution of pathological left-handedness, in brain-injured populations, will confound any relationship between handedness and cerebral dominance. Landsdell (1971) has, in fact, recently reported 18 cases of brain-injury (primarily epileptic) who, because of early left hemisphere damage, had speech dominance in the right hemisphere. Fifteen of the patients were left-handed and presumably pathological left-handed. These studies suggest (Branch, et al., 1964; Landsdell, 1971) that pathological left-handedness may represent a switch in the speech mechanisms as well as in handedness.

On the basis of the preceding comments, one might speculate as to whether the concept of pathological left-handedness is related to long-established reports

of increased variability in manual performance or preference in left-handers (Bingley, 1958; Zangwill, 1962; Palmer, 1964; Satz, Achenbach & Fennell, 1967). Although these studies have postulated genetic and cultural determinants to account for the increased manual variability in sinistrals, it is equally possible that selection factors may have increased the likelihood of sampling Ss who may have sustained minor birth injuries without clinical sequelae (e.g., epilepsy and mental retardation). If true, it would account, in part, for reports of superior right-hand dexterity in many of these Ss (Satz, et al., 1967). This possibility should again underscore the need for more information regardin the nature of the selection sample.

The preceding discussion briefly reviewed the hypotheses generated by the model and examined some implications of these hypotheses on variables or phenomena related to handedness. While brevity was sought in the formulation of the model, certain issues were excluded which should perhaps be clarified for subsequent tests of the model.

First, handedness was treated as a dichotomous variable in both the model and in the test of the model (Tables 4 and 5). As such, the classification of handedness was implicitly assumed to rest on dexterity or preference measures. Cases of mixed or ambidexterous preference, in Tables 4 and 5, were classified as left-handers largely because of previous studies which have demonstrated an sssociation between ambidexterity and left-handedness (Bingley, 1958; Satz, et al., 1967). Although handedness was classified into dichotomous categories, largely in the interest of parsimony (N. Geschwind, personal communication), it should be recognized that self-reports of hand preference, particularly in sinistrals, have not correlated well with functional dexterity of the preferred hand (Satz, et al., 1967). For this reason, the present definition of left-handedness must be viewed with caution.

A second problem concerns the definition of natural handedness. It was suggested that this term may imply inheritable determinants. While cultural factors have also been shown to shape the direction and magnitude of manual laterality (Hecaen, et al., 1964), the present definition of natural handedness (NH) is postulated to represent both genetic and cultural determinants which should be differentiated from pathological handedness (PH). Thus, manifest handedness would comprise cases of NH and PH.

A third problem is related to those individuals who become switched handers due to early brain injury contralateral to the natural hand (P_{SH} = .21). No attempt was made in the present model to specify more precisely the mechanisms (genetic or neurological) which might increase the chances of manual transfer. It is quite possible that both genotype and lesion variables are underlying determinants. Lesion area would naturally be important, particularly if there was encroachment upon the motor areas. However, there is clinical evidence which limits the accuracy of this statement--namely, those children with right-hemiparesis who still prefer their right hand for manual activities (Hecaen, et al., 1964). Consequently, one is forced to entertain other mechanisms, probably genetic, which might interact with lesion specificity in accounting for cases of manual transfer. The concept of heterozygosity (DR genotype) has already been suggested by some investigators to be related to mirror-imaging in monozygotic twins (Rife, 1955; Annett, 1964; Satz, Jones & Fennell, 1969). These authors have suggested that the DR genotype predisposes the child to more variable manual and hemispheric speech laterality after brain injury or under conditions of excessive cultural pressure. Although there is some indirect support for this position, the mechanisms still remain obscure.

It is also possible that sex factors are related to the phenomenon of manual transfer. There are reports, although indirect, which reveal a slightly higher

incidence of manifest left-handedness in boys and a corresponding increase of prematurity and brain injury in boys[4] (Hecaen, et al., 1964). However, no one has yet shown whether the ratio of boys to girls is higher in pathological handedness groups. Again, the present model is unable to specify more precisely the contribution of genotypic and lesion variables in the development of pathological handedness. Intuitively one might predict that the necessary factor is lesion specificity, assuming that the lesion occurs before the age of two. However, the probability of manual switch may then depend upon certain genotypic or sex-linked factors which interact with the lesion variables to facilitate the switch in hand preference. These variables, or some combination thereof, may thus provide a necessary and sufficient condition for transfer. A major contribution to knowledge in this area could be made if one could demonstrate differences in familial handedness or genotype between brain-injured children who switched hand preference (pathological) and those who did not. Unfortunately, advances in this area must await new developments in behavioral genetics.

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FOOTNOTES

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[3] The fact that the vast majority of the excluded sinistrals (N = 49) had speech laterllized to the right hemisphere indicates that the language mechanisms had at least shifted after early brain injury.

[4] The incidence of boys is also substantially higher than girls in early learning disability which, if due to early brain damage, might account, in part, for reports of a relationship between handedness and specific learning disability. Again, the relationship might be due to the contribution of pathological left-handedness in these children.

Table 1

Distribution of Natural Handedness Groups
Before Random Brain Lesion in Projected Sample
Where Left-Handedness is Estimated at 8 Percent.

Lesion Side

		L	R	T	%
Natural Hand	L	40	40	80	(8)
	R	460	460	920	(92)
	T	500	500	1000	
	%	(50)	(50)		(100)

Table 2

Estimated distribution of Handedness Groups (Natural and Pathological) <u>after</u> random brain lesion, assuming 21 percent shift in natural hand contralateral to lesion (P_{SH} = .21).

		Lesion Side L	R	T	%
Manifest Hand	L	40 + 97(P)	32	169	(17)
	R	363	460 + 3(P)	831	(83)
	T	500	500	1000	
	%	50	50		(100)

Table 3

Estimated distribution of Handedness Groups (Natural and Pathological) after random brain lesion, assuming 50 percent shift in natural hand contralateral to lesion (P_{SH} = .50).

		Lesion Side L	R	T	%
Manifest Hand	L	40 + 230(P)	20	290	(29)
	R	230	460 + 20 (P)	710	(71)
	T	500	500	1000	
	%	(50)	(50)		(100)

Table 4

Observed distribution of Handedness Groups (Natural and Pathological) after random brain lesions in sample of 522 adult epileptic patients prior to neurosurgery. Data abstracted and recomputed from Penfield & Roberts, 1959.

		Lesion Side			
		L	R	T	%
Manifest Hand	L	67	22	89	(17)
	R	179	254	433	(83)
	T	246	276	522	
	%	(47)	(53)		(100)

Table 5

Observed distribution of Manifest Handedness Groups in 386 adult epileptic patients after removal of Ss with early brain lesions (before age 2). Data abstracted and recomputed from Penfield & Roberts, 1959.

		Lesion Side L	R	T	%
Manifest Hand	L	18	15	33	(8)
	R	157	196	353	(92)
	T	175	211	386	
	%	(46)	(54)		(100)